1.0

4.5
5.0
5.6
6.3

2.8

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3.2

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MICROCOPY RESOLUTION TEST CHART

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PERFORMANCE AND PHYSIOLOGICAL EFFECTS OF ACCELERATION-INDUCED (+Gz) LOSS OF CONSCIOUSNESS

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>Flight profiles flown in today's aircraft routinely introduce the pilot to G levels above individual tolerances, especially with the acceleration stresses imposed by air combat maneuvering. This places the pilot in a situation where loss of consciousness (LOC) could occur with little or no warning and of which he may not be aware did occur, even after he regains consciousness and recovers the aircraft.

The objective of this study is to determine how well and how soon a pilot can regain control of an aircraft if he accidentally loses consciousness while in a high-G maneuver. A secondary objective is to determine how much warning time a pilot has from peripheral light loss (PLL) until he experiences LOC. Eight volunteers were repeatedly taken to deliberate LOC on the NAVAIRDEVCEN human centrifuge under these different G-onset conditions simulating (cont.)

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a TACAIR environment. Twenty LOC episodes occurred during the study. The period of complete incapacitation for all LOCs was a mean 12.6 seconds which, when combined with the period of confusion and disorientation immediately following recovery, results in a total mean time of 25 seconds during which the pilot is unable to adequately perform. This is more than enough time for a disaster to occur, especially in an unstable aircraft. A pilot may, during the period of partial incapacitation, misinterpret his aircraft's situation and induce a departure from controlled flight, overstress his aircraft, or unnecessarily activate the ejection mechanism. This study calls attention to the importance of training pilots for increasing their G tolerance and of making them aware of the dangers of accidental LOC.

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HOUGHTON JO, McBRIDE DK, HANNAH K. *Performance and physiological effects of acceleration-induced (+ Gz) loss of consciousness.* Aviat. Space Environ. Med. 1985; 56:956–65.

Loss of consciousness (LOC) was intentionally induced by exposing eight volunteers to individually-titrated levels of head-to-foot acceleration (+Gz) using 2- and 4-s onset rates (mean = 6.1 +Gz required to induce LOC) and a gradual, .067 G · s⁻¹ onset rate (mean = 7.2 Gz required). Subjects were trained over a prior 2-week period on a multitask battery comprising three simultaneously executed tasks representative of those required in piloting, and then centrifuged to LOC at each of the three onset rates on alternate days. Performance was assessed for 5 min prior and 7 min after each LOC. Primary results indicated: a) significant and substantial impairment in the two discrete response secondary tasks (choice reaction time and arithmetic computation), with mean recovery to pre-LOC levels within 3 min on each task, b) no group mean impairment for the primary, compensatory tracking task, c) substantial individual variation in physiologically and behaviorally defined recovery from LOC, d) a negative influence of aerobic fitness on G tolerance and LOC recoverability, and e) that recovery effects were not generally dependent upon onset rate. Mean absolute incapacitation (head dropped) for the rapid onset rates was 12.1 s. For the gradual onset rate, mean absolute incapacitation was 16.6 s. Mean relative incapacitation (head erect, no voluntary task engagement) for the rapid onset rates was 11.6 s; for the gradual onset rates, mean relative incapacitation was 15.7 s. Evidence for retrograde amnesia effects was equivocal.

LOSS OF CONSCIOUSNESS (LOC) due to reduced blood flow to the brain during head-to-foot (+Gz) acceleration is a well-known risk in high-performance aircraft (8). In order to evaluate the specific degree of risk to the pilot imparted by LOC, it is necessary, of course, to consider various aspects of performance over the entire time course of the LOC evolution. Unfortunately, however, the scarcely available information on this topic is far from sufficient, for a number of reasons. Because of the associated medical ramifications of *accidental* LOC, behavioral measures of recovery are simply not routinely taken, even in laboratory environments. The only data accumulated are typically *physiological*, and even these recordings are contaminated because of the emergency medical procedures which usually follow accidental LOC episodes. The available *behavioral* indicators of LOC recovery are largely anecdotal. Concentrating primarily on amnesia phenomena, these measures provide practically no generalizable information about the recovery of pilot skill following LOC.

Based largely on laboratory experience with accidental LOC, and on a review of the paltry archives on LOC recovery, it was decided that performance capability during three separate stages of LOC recovery deserved careful examination: a) the *absolute* incapacitation stage, where the subject is unarguably unconscious, b) the *relative* incapacitation phase—a period of disorientation and confusion which typically follows absolute incapacitation, and c) the *normalization* stage, during which the subject exhibits voluntary, goal-directed control, manages to reengage the various piloting tasks, and presumably approaches pre-LOC performance norms. This study addresses skilled

performance, as well as the physiological consequences of LOC, during these three phases of recovery.

MATERIALS AND METHODS

Seven males and one female volunteered and qualified for the study. None of the participants was a designated aviator, although flight histories varied considerably across individuals. Ages ranged from 18–39 years with a mean age of 29. All of the participants passed Class II U.S. Navy aviation physical examinations, as well as examinations of cervical, thoracic, and lumbar spine X-rays, electroencephalograms, resting and stress electrocardiograms (Bruce extended protocol), Luria Nebraska psychoneurologicals, and standard clinical blood chemistry screening protocols (Roche Laboratories No. 42). Cardiovascular fitness data, as indicated by treadmill stress testing are presented in Table I.

TABLE I. CARDIOVASCULAR FITNESS PARAMETERS ACROSS PARTICIPANTS AS INDICATED BY PERFORMANCE ON AN EXTENDED BRUCE STRESS TEST.*

	Min.	Max.	Mean	S.D.
Resting HR (B.P.M.)	60	80	72	6.7
Max HR (B.P.M.) •	174	199	185	8.6
Treadmill Time (seconds)	484	1260	795	356.0

*Oxygen uptake was not recorded. Estimates may be calculated based upon various formulae.

All subjects underwent abbreviated physical examinations immediately before and immediately after each acceleration run. Participants were monitored by a flight surgeon during each run. Monitoring equipment included a two-lead electrocardiogram, audio-video recorder, two-way voice communication system, doppler ultra-sound blood velocity sensor positioned at the superficial temporal artery, ear oximeter, respiration monitor, and intermittent systolic and diastolic blood pressure recorders. Subjects were informed verbally and in writing of potential risks according to procedures approved by the Naval Air Development Center's (NADC) Committee for the Protection of Human Subjects. Extensive follow-up medical examinations, including re-administrations of the complete pre-experimentation medical battery, revealed no untoward consequences for any of the participants.

Apparatus and Tasks

Centrifuge: Acceleration was provided by the NADC Dynamic Flight Simulator (DFS). This device has been extensively described elsewhere (3). During acceleration runs, peripheral light loss (PLL) and central light loss (CLL) were measured on a semicircular light bar as previously described (2). This procedure was altered during LOC runs so that the light bar was used only to track the extent of peripheral or central vision loss, not to stop the run or establish endpoints.

Performance Tasks: The performance tasks were chosen to emulate, superficially at least, the types of performance typically required in piloting modern aircraft. That is, information input to the subject was auditory and visual, required processing of the information was spatial and verbal, and information

output (responding) was vocal as well as nonspeech motor. The three tasks performed simultaneously were a two-dimensional compensatory tracking task, a numerical computation task, and a choice reaction time task.

The compensatory tracking task required the subject to maintain the intersection of two orthogonal CRT-displayed crosshairs within a dime-sized central target area. Each line was pseudorandomly driven from its respective center line by a force function comprised of three sine waves (1/3, 1/7, and 1/11 Hz) at amplitudes of 22.2–44.4% of the screen width or height. The subject controlled excursions of the crosshairs from horizontal and vertical centerlines by manipulating a side-mounted, pressure-sensitive joystick. Rate control was proportional to stick pressure; 5 lbs of pressure provided 110% control in 0.5 s. Performance was automatically scored and recorded as percent time on target over 1-min intervals.

The computation task required vocal response solutions to relatively simple (e.g. "8 x 7"), two-digit arithmetic problems. Values (subtrahends, addends, etc.) and operations (subtraction, addition, or multiplication) were applied randomly. The problems were presented binaurally at 10-s intervals by a pre-recorded tape. Reaction time was scored as the time elapsed from completion of the problem statement until the subject's initiation of a vocal response.

For the choice reaction time task, a red and a green jewel lamp were co-mounted 15° to the right of, and 10° above the design eye. An identical pair of lamps was mounted symmetrically to the left, and above the design eye. An Apple II computer drove singular activations of the lamps approximately every second, according to a semirandom schedule. The subject's job was to deactivate the presentations by depressing a left rudder-mounted footswitch for either of the red presentations, or a right rudder-mounted footswitch for either of the green presentations. Accuracy and mean reaction time measures were recorded by the computer for each min of performance.

Experimental Protocol

Three acceleration profiles were applied in a completely within-subjects experimental design. The first, a gradual onset rate (GOR), was a 0.067 G · s⁻¹ ramp, increasing linearily to an 8-G maximum endpoint. The two rapid onset rate (ROR) profiles were haversine-shaped onsets to predetermined G_z plateaus. The plateau level was maintained for 15 s or to the endpoint, if the desired endpoint was reached in less than 15 s. This technique was used to avoid overshooting the endpoint due to the lag from stoppage of blood flow until symptoms (vision loss or LOC) occurred. The two ROR profiles comprised a 2-s rise to plateau, and a 4-s rise to plateau, respectively. This resulted in onset rates of 2.2–3.0 G · s⁻¹ for the 2-s rise time, and 1.0–1.5 G · s⁻¹ for the 4-s rise.

To ensure reliability of performance measures, subjects were trained on the performance tasks (approximately 300 min each over a 2-week training phase) until scores leveled, and intertrial correlations approached unity (4,7). Prior to LOC runs, relaxed

G tolerance to PLL was ascertained for each subject at each of the three onset rates. The subjects were then taken to a G level at which LOC occurred at each of the three acceleration profiles. The sequence of the acceleration profiles was randomized (without replacement) for each subject. Only one run to LOC was allowed per subject in any 24-h period. The G level required to produce LOC was minimized by positioning the subject upright (13^c seatback angle) and by disallowing the use of G tolerance-enhancing maneuvers or anti-G garments.

Performance was measured for 5 min pre-LOC and 7 min after each LOC (Fig. 1). Immediately before initiation of eight of the experimental runs, the subject was assigned a runway heading (e.g. "36 right") via the intercom system. The subject immediately acknowledged and confirmed the runway assignment. This procedure was administered once to each of the participants so that assignments were made for four of the GOR runs and two each for the two ROR profiles. Precisely at the conclusion of the fifth minute of the pre-LOC segment, the performance tasks were deactivated and the compensatory tracking side stick was switched functionally so that it controlled the NADC light bar. The subject was given 30 s to stabilize PLL tracking prior to the acceleration run. Immediately before the acceleration onset, subjects were assigned a new runway heading (e.g. "27 left"), again with the instructions to acknowledge and confirm the updated assignment verbally. During the acceleration run, the subject's only task was to track peripheral vision.
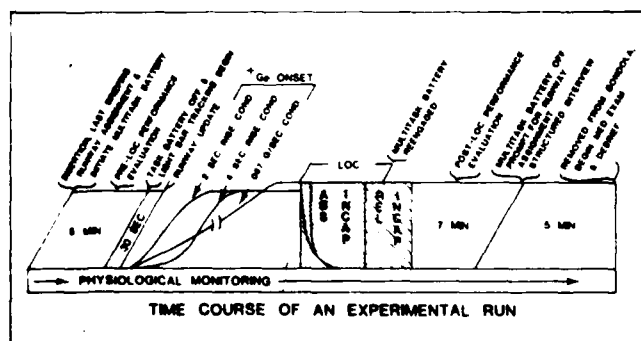

Fig. 1. Course of events for LOC runs (not scaled). Sequence depicted was used for all evolutions except that runway assignments/queries (tests for amnesia effects) were used only once per participant.

Each acceleration run was stopped when LOC occurred. Centrifugation was stopped by any of the following methods: a) if acceleration reached 8 G, a G-limiter automatically stopped the run; b) subjects maintained center position on a dead-man switch with the left hand so that either releasing at LOC or squeezing the spring-loaded trigger stopped the run; c) the medical monitor and the flight director each maintained stop buttons, activated immediately at visual signs of LOC; d) the centrifuge operator and project engineer also maintained stop buttons. The dissipation of G subsequent to all LOC episodes followed a negatively-accelerated exponential decay, so that G was

reduced to 37% of plateau at precisely 4 s after the initiation of the offset.

When LOC occurred, the performance battery was immediately energized and the subject was verbally and repeatedly prompted by the flight director to begin performing until he or she actually did so. Otherwise, communication was not permitted during the 7-min, post-LOC, performance-evaluation segment. It is difficult or impossible to predict the specific influence of prompting on the various parameters of recovery. It is plausible, if not assumed, that repeated encouragements shorten the incapacitation times associated with LOC episodes. However, there is little or no documented evidence to suggest that such prompting should systematically interact with other controlled or uncontrolled variables, such as onset profile, anthropometric characteristics, etc.

Immediately at the conclusion of the post-LOC performance phase, the subject was queried for recall of runway assignment. Non-recall of the *updated* runway assignment was interpreted as partial evidence for LOC-induced retrograde amnesia effects. An extensive, structured interview ensued. The entire LOC evolution and debrief phase were audio/video recorded.

RESULTS AND DISCUSSION

Physiological Effects

The primary questions, of course, deal with the effects of acceleration-induced LOC on skilled behavior. Of preliminary concern are considerations of the acceleration profiles required to produce LOC, and the physiological phenomena which resulted during the time course of the experimental runs. The associated data are summarized in Table II and Table III, respectively. As shown in Table II, the average ROR acceleration profile required to produce LOC was slightly greater than +6 G_z with mean durations of 15–20 s, including onset time. Not surprisingly, slightly more +G_z (mean $\cong$7G) and a longer duration at > 1 G_z were required

TABLE II. ACCELERATION PROFILES REQUIRED TO INDUCE LOC.*

	.067 G·s⁻¹	2−s Rise	4−s Rise
+G$_z$			
mean	7.2	6.1	6.3
min.	5.8	5.3	5.0
max.	8.0	7.0	7.0
S.D.	0.8	0.7	0.8
Time @ >1 G (seconds)			
mean	83.5	16.6	18.9
min.	67.0	7.0	17.0
max.	105.0	19.0	23.0
S.D.	32.1	4.3	1.5
G Density**			
mean	367.4	70.8	68.1
min.	250.1	50.1	55.5
max.	422.5	98.8	91.6
S.D.	67.8	17.2	13.7

*Centrifugation failed to induce LOC for two participants in the GOR condition and for one in the 4−s condition

**G density is the integral of G from onset, through offset to 1.02 G.

TABLE III INCAPACITATION DATA.*

	Time (sec) from onset of G to:				Total Warning Times
	Doppler Loss**	Peripheral Light Loss+	Blackout++	LOC	
0.67 G·s⁻¹					
mean	58.2	55.7	64.8	73.3	37.1
min.	27.0	37.5	41.0	51.0	30.0
max.	79.5	82.5	95.0	97.0	41.8
S.D.	17.7	14.2	21.1	33.0	5.6
2-s rise					
mean	4.0	6.5	7.0	9.2	4.1
min.	1.0	2.5	2.6	8.2	2.0
max.	8.0	10.5	10.8	12.0	8.1
S.D.	2.7	2.3	2.4	1.4	2.2
4-s rise					
mean	3.8	6.8	6.6	10.7	5.0
min.	3.0	4.0	4.0	9.0	1.5
max.	4.3	8.8	8.9	14.5	7.6
S.D.	0.5	1.9	2.0	1.9	2.2

* A lack of precision here results from variability in reaction time, and the difficulty of both maintaining the hand controls under G and in establishing peripheral vision.

** Loss of doppler ultrasound blood velocity indication at the superficial temporal artery, indicating loss of cranial blood flow

+ PLL (re: 60° central angle) as measured by tracking on the light bar.

++ Loss of PLL and CLL before LOC

§ Time elapsed from PLL to LOC.

to induce LOC for the GOR profiles. As can be seen in Table III, the warning times (PLL to LOC) in the ROR conditions were much shorter than in the GOR conditions. The 4- to 5-s warning times for ROR provide ample opportunity for LOC avoidance, but the relatively long (mean ≅37 s) and variable (S.D. ≅6 s) warning times associated with GOR suggest that PLL is probably not a very reliable premonitory cue for LOC under slow onset conditions. Note that a maximum onset of approximately 3 G·s⁻¹ was attained in this study. Since current tactical aircraft are capable of producing much higher onset rates, substantially shorter warning times would be expected in operational contexts.

Absolute incapacitation is defined presently as the time elapsed from head drop to the point at which the head is raised and maintained in an upright orientation. Since this is somewhat subjective, three judges— one flight surgeon, one aviation physiologist, and one aerospace experimental psychologist—provided multiple estimates of incapacitation duration for each video-recorded LOC episode. Inter-rater reliability (correlation) coefficients exceeded 0.90. Mean absolute incapacitation and standard deviations for the three acceleration profiles are depicted in Fig. 2. The incapacitation times shown here are within the range of previously reported accidental LOC episodes (1,9). Mean absolute incapacitation for the combined ROR conditions was 12.1 s. For GOR, mean absolute incapacitation was 16.6 s. The differences among the three means (two ROR, one GOR) for the three G-
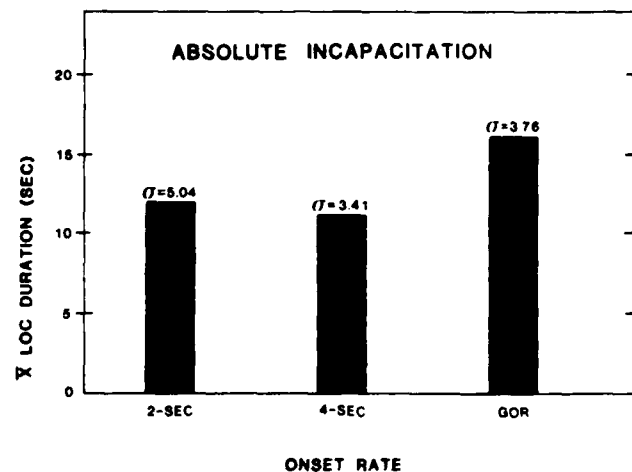

Fig. 2. Means and dispersions of absolute incapacitation durations for the three onset conditions. The means are not statistically different.

onset conditions are not statistically reliable [F (2.11) <1].

Total incapacitation is defined as the time elapsed from head drop to the initiation of the first purposeful, voluntary response with the joystick. The same judges were empaneled and inter-rater reliabilities were, again, quite high (rs > 0.90). Total incapacitation thus comprises absolute and relative incapacitation, the latter defined as the time from the initial maintenance of head posture to the commencement of voluntary limb manipulation. Relative incapacitation was described by all subjects during debrief as one in which consciousness was returning, although purposeful behaviors were difficult or impossible to integrate. Mean relative incapacitation for the combined ROR profiles was 11.6 s; for GOR, mean relative incapacitation was 15.7 s. Differences among means are not statistically reliable [F (2,11) < 1]. Mean total incapacitation times are portrayed in Fig. 3. For the combined ROR conditions, mean total incapacitation was 23.7 s. For GOR, mean total incapacitation was 32.3 s. The differences among the three means are not statistically reliable [F (2,11) < 1].
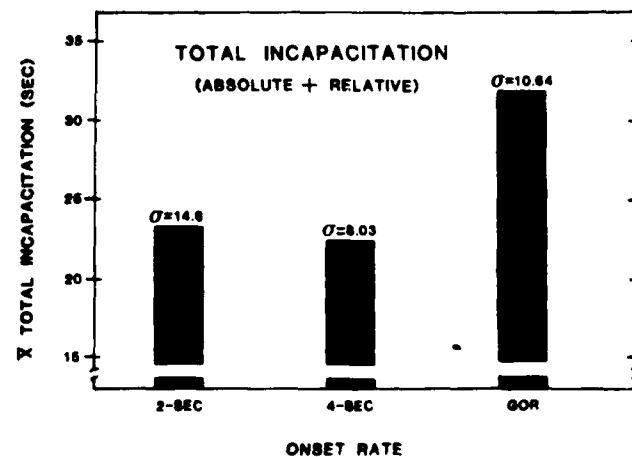

Fig. 3. Means and dispersions of total (absolute + relative) incapacitation times for the three onset conditions. The means are not statistically different.

4

Total incapacitation times should be interpreted as conservative here since a) the subjects were continually prompted to regain control, b) LOC was induced at G levels probably lower than those associated with accidental LOC in flight (i.e. no anti-G garments or straining maneuvers were employed here), and c) the LOCs were not accidental (i.e. forewarning that LOC is impending may provide the subject with some presently unspecifiable preparation that could serve to reduce the intensity or duration of incapacitation, especially relative incapacitation).

Systolic and diastolic pressures as recorded across the pre- and post-LOC segments are shown in Fig. 4, as are systolic rates in Fig. 5. Perhaps the most significant characteristic of these depictions is the apparent anticipatory effect associated with the pre-acceleration portion of the runs.

Performance Effects

The performance consequences of LOC are considered from two perspectives. First, we examine and discuss group mean effects. Second, we turn to a

treatment of individual variation in G tolerance and in the various parameters of LOC recovery.

Group Mean Effects: Due to the relatively small sample employed for this study, the replicability of differential effects of acceleration profile (i.e. GOR vs. ROR) were intentionally ignored. An inspection of Fig. 6, 7, and 8 confirms that even if differential effects were found to be reliable, the magnitudes of these differences relative to the overall effects of LOC would
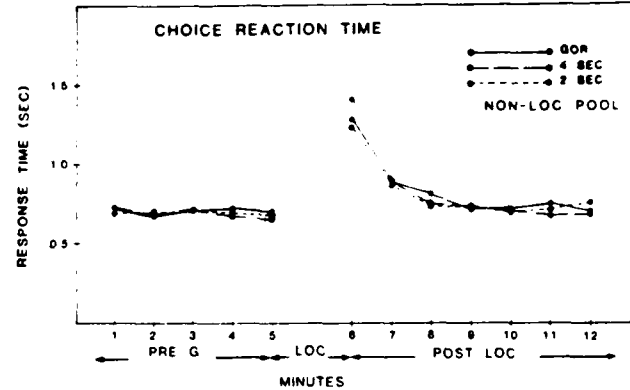
Fig. 6. Mean choice reaction time scores suggest little or no effect for onset type. However, post-LOC reaction time for all onset rates is, temporarily, double that of pre-LOC baseline levels. There is little or no effect for the pooled - G, non-LOC data.

Fig. 4. Mean systolic and diastolic blood pressures as recorded during pre- and post-LOC segments. Note the apparent anticipatory effect.
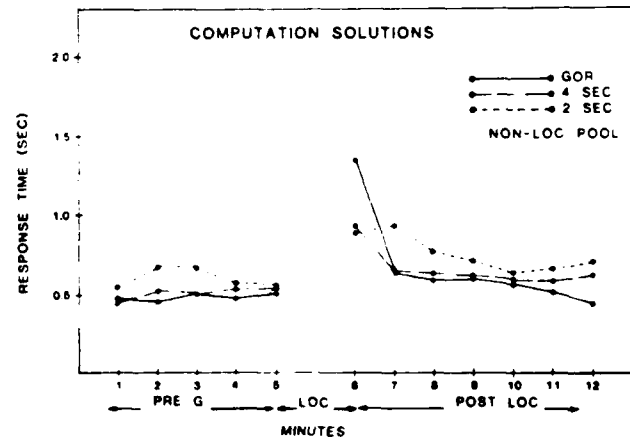
Fig. 7. Mean response times for the computation task. As for the choice reaction time task, onset rate produces no differential effect, and mean response time roughly doubles for at least 60 s into the post-LOC segment.
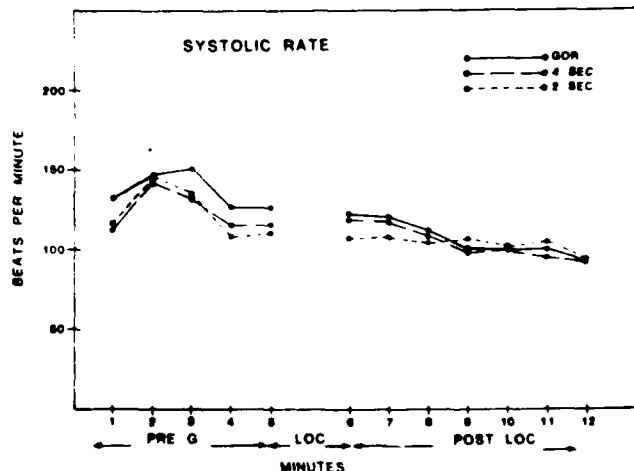

Fig. 5. Mean systolic rates, pre- and post-LOC. Here, as with BP, there is an apparent anticipatory effect.

Fig. 8. Mean compensatory tracking accuracies. Neither onset rate nor LOC produces reliable effects on time-on-target scores.

almost certainly be trivial. Alternatively, and in order to maximize the statistical power of the tasks employed, performance scores for each of the three tasks were averaged within trials, across G-onset profiles for each subject. This technique serves to smooth performance trends and reduce within-subject error variance.

A subjects × treatments analysis of variance revealed a replicable time-course effect for choice reaction time performance at $F = 18.33$ (11,77) and $p < 0.001$. Fig. 6 suggests that performance is degraded during the first minute of recovery, and that it progressively improves in 2–3 min to approximating pre-LOC levels. Planned t tests for correlated samples confirmed that performance is significantly impaired during minute 6 effect [$t(7) = 5.50$, $p < 0.005$] compared to the pooled scores from minutes 1–5. Thereafter, choice reaction time performance is not reliably distinguishable from that associated with pre-G levels.

The same trend holds for the computation task [$F(11,77) = 5.32$, $p < 0.001$]. The degradation, relative to the minute 1–5 pool, is reliable for minute 6 [$t(7) = 2.33$, $p < 0.05$] and min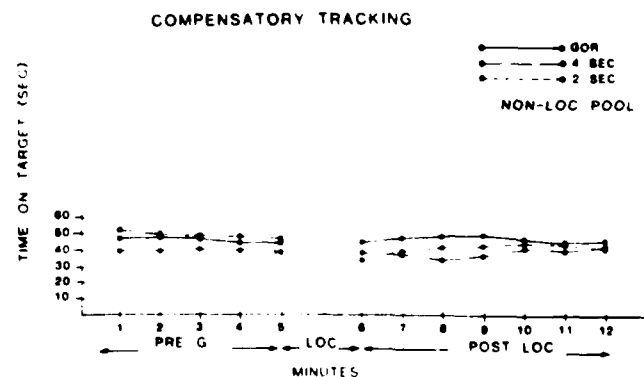ute 7 [$t(7) = 3.47$, $p < 0.01$], but not for any subsequent 60-s segment ($t < 1.0$). Here, as for choice reaction time, the magnitude of the minute 6 effect is roughly a doubling of baseline reaction times.

Turning to compensatory tracking, a somewhat surprising outcome is revealed. Here, there simply is no time course effect [$F (11,77) < 1$]. In laboratory settings where continuous practice is experimentally interrupted with a rest period of a few seconds or more, as in the present case, there is typically an increase in the quality of response in the immediate post-rest phase relative to pre-rest stages (6). This so-called reminiscence effect is observed more typically in task responses which are continuous (as in tracking) rather than in intermittent responses (as in choice reaction time and computation). It may be that, in the present case, the deleterious effects of LOC have cancelled the otherwise-potentiating effects of interpolated rest. In other words, LOC might very well produce a temporary, debilitating effect on tracking proficiency. The effect is masked in the present case, however, by the positive effect of temporary task disengagement. We are, therefore, left with an interpretive puzzle. That is, should the apparent non-effect of LOC on tracking be regarded as

the insensitivity of a *primary* task to LOC, or as the insensitivity of a *continuous* task to LOC, or both, or neither? The answer simply cannot be adduced from the present findings.

The *time course* effect has been so termed because one cannot determine the specific effects of LOC on performance independently of the effects of G exposure, blackout, time interpolated between pre- and post-sessions, etc. However, performance data were recorded for six experimental runs during which acceleration failed to render the subject unconscious, and for seven runs where acceleration endpoints were being titrated. All elements of the experimental runs were common to all of the non-LOC runs, except for the production of LOC. The performance data from all non-LOC runs were subsequently pooled without regard to acceleration profile and subjects × treatments variance analysis was applied. The procedure revealed no reliable time course effect (F < 1.0) for any of the three performance measures. An inspection of the minutes 5 and 6 non-LOC means, as indicated in Fig. 6, 7, and 8, suggest this null result. Although these findings are not conclusive in establishing that the performance decrements associated with LOC are *purely* LOC-specific, the data strongly suggest that at least *some* portion of the performance decrement is LOC-dependent.

The magnitudes of the effects reported here are similar to those found in a study (5) in which trained subjects were required to engage a large battery of some 20 discrete response tasks immediately on being awakened from various stages of sleep. There, although there was no primary task *per se*, and none of the tasks required continuous responses, reaction time generally suffered significantly across tasks during the first minute of post-awakened testing. Skill progressively returned to near normal baseline levels within 4–5 min.

Evidence for retrograde amnesia in the present study is, at best, equivocal. The updated runway 7 min after initial recovery, was correctly recalled by 5 of the 8 subjects [χ^2 (1) < 1, where it is assumed that control condition (no G or LOC) recall is perfect]. This does not mean, of course, that amnesia effects are not produced by LOC. Unawareness of, or inability to recall, the LOC event has been reported in *accidental*

TABLE IV. CHOICE REACTION TIME OF INTERTRIAL CORRELATIONS.*

Min.	Pre–LOC				Post–LOC						
	2	3	4	5	6	7	8	9	10	11	12
1	.90	.93	.95	.96	.53	.78	.83	.89	.89	.89	.80
PRE- 2		.95	.91	.87	.53	.85	.92	.89	.94	.88	.91
3			.90	.92	.55	.84	.92	.91	.92	.95	.89
LOC 4				.87	.63	.75	.86	.90	.87	.89	.80
5					.57	.78	.87	.88	.91	.91	.83
6						.25	.66	.63	.62	.54	.56
7							.41	.71	.78	.77	.58
POST- 8								.93	.96	.94	.96
9									.92	.95	.93
LOC 10										.96	.94
11											.95

* Mean r for all three acceleration profiles using Fisher r to z to r transforms.

TABLE V. COMPUTATION OF INTERTRIAL CORRELATIONS *

	Min	2	3	4	5	6	7	8	9	10	11	12
		Pre-LOC				Post-LOC						
	1	.93	.80	.68	.72	.22	.56	.57	.77	.82	.61	.47
PRE-	2		.85	.83	.77	.46	.72	.71	.84	.90	.78	.65
	3			.93	.85	.34	.51	.63	.69	.63	.66	.60
LOC	4				.80	.43	.53	.70	.67	.63	.59	.66
	5					.48	.61	.65	.75	.73	.83	.71
	6						.78	.84	.59	.60	.79	.84
	7							.62	.67	.68	.78	.71
POST-	8								.67	.78	.83	.85
	9									.82	.84	.74
LOC	10										.76	.91
	11											.84

* Mean r for all three acceleration profiles using Fisher r to z to r transforms.

TABLE VI. COMPENSATORY TRACKING OF INTERTRIAL CORRELATIONS *

	Min.	2	3	4	5	6	7	8	9	10	11	12
		Pre-LOC				Post-LOC						
	1	.91	.78	.90	.83	-.24	-.06	-.13	-.10	-.44	-.42	-.37
PRE-	2		.86	.89	.89	-.29	-.12	-.09	-.17	-.55	-.49	-.40
	3			.94	.96	-.10	.00	.02	.03	-.35	-.26	-.26
LOC	4				.95	-.17	-.09	-.15	-.13	-.58	-.44	-.31
	5					-.18	-.07	-.05	-.07	-.36	-.29	-.37
	6						.90	.88	.91	.75	.74	.69
	7							.89	.96	.71	.69	.48
POST-	8								.95	.78	.77	.65
	9									.87	.81	.70
LOC	10										.99	.92
	11											.94

* Mean r for all three acceleration profiles using Fisher r to z to r transforms.

LOC episodes (9). This inability to recall was not replicated in the present study, perhaps because the subjects were aware that LOC was to occur or because there were familiar sound and motion cues to indicate that time and events had passed. Nevertheless, in 3 of the 21 LOC episodes, the subject was reluctant to believe that LOC had occurred until seeing the LOC videotape.

Individual Variation: Individual variation in the three performance tasks, as indicated by intertrial correlations, is depicted in Tables IV, V, and VI. The trends are generally typical of those reported in studies of skill (4,7), i.e. there is apparent obedience to laws of single tetrad differences and superdiagonal form. The intercorrelation patterns here, however, suggest that there is substantial inter-subject variability in rates of LOC recovery. This is particularly interesting for compensatory tracking; there was no group mean effect of LOC on this aspect of skilled performance. However, the nonsignificant intercorrelations of pre- to post-LOC tracking scores indicate that individual reactivity to LOC was very high. In other words, although group performance was not affected on balance by LOC induction, individual recovery was highly differential.

In order to explore more fully the individual patterns in LOC recovery, scores for the 10 physiological parameters associated with producing and recovering from LOC were combined across onset profiles for each participant and subjected to a varimax rotation factor-analysis procedure. The 45 interparameter correlations are presented in Table VII. The analysis yielded two primary factors. An inspection of the factor loadings in Table VIII suggests that the two factors are, perhaps, representative of the clusters of variables associated with G tolerance, and LOC recoverability. That these factors are orthogonal is interesting, for this suggests that one's ability to recover physiologically from LOC is not related to one's unaided (i.e. no straining maneuvers, etc.) ability to tolerate the LOC-inducing properties of $+G_z$. This finding, considered especially in light of the differential rates of performance recovery, prompted an in-depth examination of individual variation i performance and, specifically, of the relationships between performance measures and the various indicants of G tolerance and LOC recovery.

An examination of the correlation coefficients in Table IX suggests that cardiovascular fitness is proportional to blood O_2 saturation, as estimated by the ear oximeter during the experimental runs. More specifically, the positive correlations between O_2 levels and the treadmill time suggest that the more physically fit individuals show greater saturation before and during the LOC episode, and for at least 45 s after

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TABLE VII. INTER-CORRELATIONS AMONG LOC-INDUCTION
AND RECOVERY PARAMETERS.

	2	3	4	5	6	7	8	9	10
1. Max. G	.62	.64	.56	.67	.68	.50	-.00	-.44	.49
2. Time @ >1 G		.99	.95	.98	.96	.88	.28	- .02	.50
3. G density (see Table II)			.94	.96	.94	.87	.27	-.00	.51
Time elapsed from G onset to:									
4. Doppler Loss				.93	.92	.83	.11	-.02	.39
5. PLL					.98	.83	.23	-.09	.53
6. Blackout						.83	.18	-.16	.46
7. LOC							.26	.07	.54
Total elapsed time:									
8. Absolute LOC								.55	.54
9. Total LOC									.15
During G/LOC:									
10. Max. Heart Rate									

TABLE VIII   ROTATED FACTOR MATRIX (TWO-FACTOR MODEL).*

	Variable	Factor 1 (G Tolerance)	Factor 2 (LOC Recovery)
1.	Max. G	.71	-.37
2.	Time @ >1 G	.98	.02
3.	G density	.97	.04
4.	G onset to doppler loss	.93	-.04
5.	G onset to PLL	.98	-.03
6.	G onset to Blackout	.97	.10
7.	G onset to LOC	.89	.11
8.	Absolute LOC duration	.27	.80
9.	Total LOC duration	-.07	.88
10.	Max. heart rate during G	.60	.39

*Loadings are Pearson r; they are indicative of the strength of the alliance of the associated variable (1-10) with the indicated factor (1,2).

TABLE IX. INTERCORRELATIONS BETWEEN TREADMILL TIMES
AND O₂ SATURATION LEVELS.

Blood O₂ Saturation (ear oximeter)	Treadmill Time
Prior to G	.64*
G onset	.83**
G offset	.80**
Sec after G offset:	
5	.78**
10	.78**
15	.85**
20	.81**
25	.82**
30	.71*
35	.89**
40	.85**
45	.79**

*$p < 0.05$, **$p < 0.01$

recovery begins. That O_2 saturation is greater for those who are more fit is, of course, not necessarily surprising. However, an inspection of the correlation figures comprising Tables X and XI suggests that participants who are more aerobically fit show inferior G-tolerance, and that aerobically fit individuals recover from LOC more slowly! Furthermore, as Tables XII,

XIII, and XIV show, recovery for several aspects of skilled performance—most notably for the primary task, compensatory tracking—is also slower for the more fit.

Just why aerobic fitness should be an apparently debilitating factor in G-tolerance and LOC recovery is not understood. Perhaps vascular elasticity is a moderating variable. Although a theoretical address

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TABLE X. INTERCORRELATIONS BETWEEN TREADMILL TIMES AND G TOLERANCE PARAMETERS

	Treadmill Time
Max G	-.52
Time at >1 G	-.62*
G density	-.78*
G onset to DI	-.33
G onset to PLL	-.60*
G onset to BO	-.45
G onset to LOC	.02

*p < 0.05

TABLE XI. INTERCORRELATIONS BETWEEN TREADMILL TIMES AND LOC RECOVERABILITY MEASURES

	Treadmill Time
Absolute LOC	.36
Total LOC	.36
Max heart rate	-.16

TABLE XII. INTERCORRELATIONS BETWEEN TREADMILL TIMES AND POST-LOC COMPENSATORY TRACKING PERFORMANCE

Min. of LOC Recovery	Treadmill Time
1	-.90**
2	-.95**
3	-.81*
4	-.93**
5	-.91**
6	-.91**
7	-.88**

*p < 0.05, **p < 0.01

TABLE XIII. INTERCORRELATIONS BETWEEN TREADMILL TIMES AND POST-LOC CHOICE REACTION TIMES

Min. of LOC Recovery	Treadmill Time
1	.15
2	.01
3	-.01
4	-.05
5	.13
6	.01
7	-.05

TABLE XIV. INTERCORRELATIONS BETWEEN TREADMILL TIMES AND POST-LOC COMPUTATION PERFORMANCE

Min. of LOC Recovery	Treadmill Time
1	.12
2	.23
3	.87*
4	-.05
5	.50*
6	-.24
7	.17

*p < 0.05

is certainly called for, such is beyond the scope of this report. Extensive examinations of the many relationships among aerobic fitness parameters and the various indicators of LOC recovery are indicated.

SUMMARY

●Acceleration required to produce LOC with no straining maneuvers, anti-G garments, etc., ranged from 5.0 to 8.0 +G_z (mean = 6.5) across ROR and GOR conditions.

●Time elapsed at > 1 G ranged from 7.0–23.0 s (mean = 17.6 s) for ROR; from 67–105 s (mean = 83.5 s) for GOR.

●Approximately 70 units of +G' (+G_z integrated over exposure time) were needed to cause LOC to occur under ROR conditions; +G_z' required under the slow onset conditions was approximately five times greater.

●Warning times—elapsed time from peripheral visual loss (re: 60° central subtense) to onset of LOC—ranged considerably across individuals (1.5–8.1 s, mean = 4.5 s) under ROR conditions. Warning times recorded under GOR circumstances were exceedingly long and variable between and within subjects. Peripheral visual loss is thus not considered to be effective as a premonitory cue under slow G-onset conditions.

● Mean absolute incapacitation for ROR conditions was 12.1 s; for GOR runs, mean absolute incapacitation was 16.6 s.

● Mean total incapacitation was 23.7 s for ROR, 32.3 s for GOR.

● Although the incapacitation durations for GOR consistently exceeded those for ROR, the differences among respective means were not found to be statistically reliable.

● Normalization, from onset of post-LOC multi-task engagement to the establishment of pre-G baseline proficiency, was almost immediate for the tracking task. Normalization for the secondary tasks varied across individuals and onset conditions within an approximate 2–3 min range. One interpretation assumes that the subject manages a finite pool of cognitive resources in order to support the simultaneous execution of the three tasks. The disruptive event of LOC reduces the depth or breadth of this pool. On recovery, then, the expenditure of resources sufficient to sustain a high level of tracking accuracy subtracts from those available for a high level of performance on secondary tasks. Generalizing to the cockpit, these findings suggest that a recovering pilot's abilities under complex perceptual-motor requirements, as are common under high-G environments, probably do not normalize for 2–3 min after recovery from LOC begins.

● Amnesia effects were not replicated. The inability to demonstrate forgetting in the present study may be attributable to few subjects, forewarning effects, or the delay strategy used to query for information provided immediately before G onset. That is, if the amnesia effect is, in fact, only temporary, the paradigm used may have been insensitive.

● Individuals varied widely in their rates of recovery. Differential effects were pronounced along most of the physiological and behavioral parameters observed.

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• Tolerance to G. and the constellation of physiological and anthropometric variables associated with involuntary G resistance, were found not to co-vary with LOC recoverability, nor with the physiological/anthropometric variables subsumed by LOC recoverability. Thus, G tolerance and LOC recoverability are independent factors.

• Although no explanation is offered, statistically reliable negative correlations among several aerobic fitness parameters and various G-tolerance and LOC-recoverability measures are reported. Follow-on experimentation is required. That aerobic fitness may detract from a pilot's ability to perform in a hostile G environment is entirely inconsistent with prevailing approaches to aircrew training.

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